Is there evidence for fascial adhesions caused by crosslinks?

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BACKGROUND: In the Fascial Distortion Model (FDM) of Typaldos D.O., one of the described fascial distortions goes along with the formation of adhesions caused by mal attached crosslinks in the banded fascia of tendons [1].
APROACH: Review of Literature. Collagen is the main element which assures the stability of connective tissue. The soundness in between the collagenic molecules is guarantied by the development of hydrogen bridges and covalent bonds called crosslinks. With aging non specific crosslinks occur by accumulation of glucose [2]. A reaction between amino acids and deoxidized sugar forms a compound (Maillard reaction) which leads to an accumulation of "advanced glycation end products" (AGEs) [3].Through aging and/or a higher level of glucose (e.g. diabetes) the unspecific crosslinks accumulate and the AGEs increase in the tissue [4, 5, 6]. It was proven that sinews which are fortified by this complex of sugar and amino acid can take a higher burden but on the other side they become stiffer [5].A characteristic of these crosslinks formed by AGEs is a decrease of solubility and a higher resistance against proteolytic decomposition [7, 8].It is known that oxidative stress advances the formation of AGE pentosid crosslinks [6, 9].Several surveys have shown that an abnormal microcirculation leads to a local hypoxia of muscle tissue. The AGEs thereby induce the protein crosslinks which in turn reduce the elasticity. [10]. When a sinew is stretched its elongation is greater than the possible lengthening of the single collagenic filament. This means that there must be a gliding between the filaments [11]. This sliding movement is hampered by non-specific cross links. By repeated, unaccustomed or excessive force the fibrils tear. With horses, fibrils with a smaller diameter were found in the core of the sinew after prolonged burden. This causes a partial looseness of the fibril bond [12]. It seems to be proven that after smaller mechanical injuries the healing of sinew is accompanied by a higher level of collagen type III synthesis. The new fibrils are significantly thinner and accordingly more prone to rupture. The proceedings leading to this collagenic build-up at the ruptured area seem to be of much older origin than the acute tearing [11].Riley observed the increase of collagen type III in cases of tendonitis of the supraspinatus sinew [13].Changes of the extracellular matrix (EMC) may be caused by intrinsic factors like alteration of cell activity or extrinsic factors like overload, repeated overexpansion and micro traumata. The newly-created collagen netting after trauma is of inferior quality. Bank et al conclude that the ECM, observed in cases of tendonitis of the supraspinatus sinew, is the result of an uncontrolled healing process in which a carefully built and highly functional matrix of the sinew is exchanged by lesser and unorganized tissue [2].
RESULT: There is evidence for the existence of adhesions caused by crosslinks.
CONCLUSION: Typaldos description of fascial adhesions matches the observations of ECM changes in cases of AGEs increase and tendonitis.

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